

**CLINICAL MANIFESTATIONS AND LABORATORY DIAGNOSIS OF
BRONCHOPULMONARY DYSPLASIA IN NEWBORN INFANTS**

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Annotation: Bronchopulmonary dysplasia (BPD) is a chronic lung disease that primarily affects premature newborns who have received prolonged oxygen therapy or mechanical ventilation. The aim of this article is to analyze the clinical manifestations and laboratory diagnostic methods of BPD in neonates. Early diagnosis of BPD is crucial for improving outcomes and minimizing complications. The study highlights key clinical signs such as respiratory distress, oxygen dependency, and poor weight gain, as well as the role of chest radiography, blood gas analysis, and inflammatory markers in establishing the diagnosis. Understanding the clinical-laboratory profile of BPD facilitates timely intervention and guides treatment strategies, ultimately contributing to better prognosis in affected infants.

Keywords: Bronchopulmonary dysplasia, newborn, prematurity, respiratory distress, oxygen therapy, mechanical ventilation, clinical signs, laboratory diagnosis, blood gas analysis, neonatal care.

Introduction.

Bronchopulmonary dysplasia (BPD) is one of the most common and challenging chronic pulmonary diseases seen in neonatology, primarily affecting premature infants, particularly those born before 32 weeks of gestation and with very low birth weight. BPD develops as a result of lung immaturity combined with various postnatal insults, such as prolonged mechanical ventilation, high concentrations of oxygen, infections, and inflammation. These factors contribute to arrested lung development, impaired alveolarization, and abnormal vascular growth, ultimately resulting in long-term respiratory complications. Despite advancements in perinatal care, the incidence of BPD has not significantly declined due to increased survival rates of extremely preterm infants. Therefore, early recognition and diagnosis of BPD are critical for implementing timely therapeutic interventions aimed at minimizing respiratory morbidity and improving quality of life. The clinical course of BPD is often characterized by persistent oxygen dependency beyond 28 days of life, tachypnea, retractions, and recurrent episodes of respiratory distress. Laboratory diagnostics play an important role in confirming BPD and differentiating it from other pulmonary conditions in neonates. Key investigations include blood gas analysis, markers of inflammation, imaging techniques such as chest X-rays, and in some cases, echocardiography to assess associated pulmonary hypertension. This article provides a comprehensive overview of the clinical and laboratory characteristics of bronchopulmonary dysplasia in newborn infants, aiming to enhance understanding and support improved clinical management of this vulnerable patient population.

Main Body.

1. Etiology and Risk Factors

Bronchopulmonary dysplasia (BPD) is a multifactorial disease that results from a combination of prenatal and postnatal factors. Major risk factors include prematurity, low birth weight, prolonged mechanical ventilation, supplemental oxygen therapy, intrauterine growth restriction, and perinatal infections. The immature lungs of preterm infants are highly susceptible to injury, and the use of life-saving respiratory support can paradoxically contribute to lung damage. Antenatal exposure to inflammation or chorioamnionitis and postnatal sepsis also play critical roles in the development of BPD.

2. Pathophysiology

The hallmark of BPD is impaired alveolar and pulmonary vascular development. In classical BPD, seen in older infants, lung injury led to fibrosis and inflammation, whereas the "new" BPD observed in extremely preterm infants is characterized by fewer and larger alveoli with minimal fibrosis. Inflammatory processes, oxidative stress from oxygen toxicity, volutrauma and barotrauma from mechanical ventilation, and disrupted signaling for normal lung growth all contribute to disease progression.

3. Clinical Manifestations

Clinical signs of BPD vary depending on the severity of the condition. Typically, BPD is suspected in infants who remain dependent on supplemental oxygen for more than 28 days

after birth. Common symptoms include: Persistent tachypnea and respiratory distress. Intercostal and subcostal retractions, Nasal flaring and grunting, Poor weight gain, Cyanosis or desaturation episodes. Increased susceptibility to respiratory infections. BPD is classified based on the severity at 36 weeks postmenstrual age (PMA) or discharge: Mild: breathing room air at 36 weeks PMA Moderate: need for <30% oxygen. Severe: need for $\geq 30\%$ oxygen and/or positive pressure support

4. Laboratory Diagnosis.

Accurate and early diagnosis of BPD relies on a combination of clinical criteria and laboratory assessments: Blood Gas Analysis: Hypoxemia and hypercapnia are common findings, with respiratory acidosis in severe cases. Complete Blood Count (CBC): Elevated white blood cell count may indicate ongoing inflammation or infection. C-reactive Protein (CRP) and Procalcitonin: Useful for ruling out concomitant sepsis or systemic inflammatory response. Chest Radiography: Provides visual confirmation of lung changes. Classic findings include hyperinflation, atelectasis, and areas of fibrosis or cystic changes. Pulse Oximetry: Monitoring oxygen saturation helps assess the need for supplemental oxygen and track disease progression. Echocardiography: Often used to evaluate pulmonary hypertension, which is a frequent complication of moderate to severe BPD.

5. Differential Diagnosis

It is important to differentiate BPD from other neonatal respiratory disorders such as neonatal pneumonia, congenital lung malformations, transient tachypnea of the newborn, and meconium aspiration syndrome. A thorough clinical evaluation combined with laboratory and imaging findings is essential for accurate diagnosis.

6. Management Overview (Brief)

Although the focus of this article is diagnosis, it is worth noting that BPD management involves a multidisciplinary approach. Supportive care, judicious use of oxygen, optimization of nutrition, use of diuretics, corticosteroids in select cases, and prevention of infections are central to therapy. Long-term follow-up is also critical to address developmental, respiratory, and neurological outcomes.

Conclusion:

Bronchopulmonary dysplasia remains a significant cause of morbidity among preterm infants, especially those requiring prolonged respiratory support. Understanding its multifactorial etiology and recognizing early clinical and laboratory indicators are essential for prompt diagnosis and effective management. Persistent oxygen dependency, respiratory distress, and poor postnatal growth are key clinical signs, while laboratory assessments such as blood gas analysis, inflammatory markers, and chest imaging play a pivotal role in confirming the diagnosis. Timely identification of BPD enables early intervention strategies that can improve respiratory outcomes, reduce complications, and enhance the quality of life for affected neonates. Continued research and advancements in neonatal care are necessary to further reduce the incidence and severity of BPD in vulnerable populations.

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